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FILE LAST UPDATED: 1 Dec 2008 (20081201/ED)

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=> s anti dlk antibody
523064 ANTI
11 ANTIS

```
      523071 ANTI
             (ANTI OR ANTIS)
         198 DLK
           1 DLKS
         199 DLK
             (DLK OR DLKS)
      339332 ANTIBODY
      408617 ANTIBODIES
      540130 ANTIBODY
             (ANTIBODY OR ANTIBODIES)
L1         2 ANTI DLK ANTIBODY
             (ANTI(W)DLK(W)ANTIBODY)

=> s anti dlk
      523064 ANTI
          11 ANTIS
      523071 ANTI
             (ANTI OR ANTIS)
         198 DLK
           1 DLKS
         199 DLK
             (DLK OR DLKS)
L2         2 ANTI DLK
             (ANTI(W)DLK)

=> d L1 bib abs 1-2

L1   ANSWER 1 OF 2  CAPLUS  COPYRIGHT 2008 ACS on STN
AN   2005:493692  CAPLUS
DN   143:39112
TI   Detection of expression level of human dlk gene for diagnosis of liver
     cancer and the use of anti-Dlk antibody for
     treatment of cancer
IN   Nakamura, Koji; Anzai, Hiroko; Yanai, Hiroyuki; Miyajima, Atsushi
PA   Kanagawa Academy of Science and Technology, Japan
SO   PCT Int. Appl., 52 pp.
     CODEN: PIXXD2
DT   Patent
LA   Japanese
FAN.CNT 1
     PATENT NO.         KIND   DATE       APPLICATION NO.      DATE
     ---------------    ----   --------   -------------------  --------
PI   WO 2005052156      A1     20050609   WO 2004-JP17499      20041125
       W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
           CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
           GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
```

LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

CA 2552553 A1 20050609 CA 2004-2552553 20041125
EP 1702982 A1 20060920 EP 2004-819413 20041125
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK, IS
US 20080112956 A1 20080515 US 2007-580567 20070430
PRAI JP 2003-399331 A 20031128
JP 2003-401585 A 20031201
JP 2003-423237 A 20031219
WO 2004-JP17499 W 20041125

AB This invention provides a method of detecting liver cancer and a novel remedy for cancer having an excellent anticancer effect. The expression of the dlk gene can be assayed by an immunoassay with the use of anti-dlk antibody or an assay of mRNA of the dlk gene. The remedy for cancer contains, as the active ingredient, an antibody undergoing an antigen-antibody reaction with Dlk expressed on caner cell surface and exhibiting an anticancer effect on the cancer cells.

RE.CNT 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2001:717804 CAPLUS
DN 135:271301
TI Myelodysplastic syndrome diagnosis with Dlk gene expression DNA microarray analysis
IN Aino, Hiroyuki
PA Kirin Brewery Co., Ltd., Japan
SO Jpn. Kokai Tokkyo Koho, 15 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI JP 2001269174	A	20011002	JP 2000-85153	20000324

PRAI JP 2000-85153 20000324

AB A method and reagent kits for diagnosis of myelodysplastic syndrome (MDS)

using Dlk gene expression as marker, are disclosed. Use of anti -Dlk antibodies for immunodiagnosis and therapy of MDS is claimed. Use of DNA microarrays and 2 dimensional electrophoresis for diagnosis is also claimed. AC133 or CD34 are used as cell surface markers. Myelodysplastic syndrome (MDS) is a slowly progressing hematol. malignancy assocd. with a poor outcome. Despite the relatively high incidence of MDS in the elderly, differentiation of MDS from de novo acute myeloid leukemia (AML) still remains problematic. Identification of genes expressed in an MDS-specific manner would allow the mol. diagnosis of MDS. Toward this goal, AC133 surface marker-pos. hematopoietic stem cell (HSC)-like fractions have been collected from a variety of leukemias in a large-scale and long-term genomics project, referred to as "Blast Bank," and transcriptome of these purified blasts from the patients with MDS were then compared with those from AML through the use of oligonucleotide microarrays. A no. of genes were shown to be expressed in a disease-specific manner either to MDS or AML. Among the former found was the gene encoding the protein Delta-like (Dlk) that is distantly related to the Delta-Notch family of signaling proteins. Because overexpression of Dlk may play a role in the pathogenesis of MDS, the disease specificity of Dlk expression was tested by a quant. "realtime" polymerase chain reaction anal. Examn. of the Blast Bank samples from 22 patients with MDS, 31 with AML, and 8 with chronic myeloid leukemia confirmed the highly selective expression of the Dlk gene in the individuals with MDS. Dlk could be the first candidate mol. to differentiate MDS from AML. The proposal is made that microarray anal. with the Blast Bank samples is an efficient approach to ext. transcriptome data of clin. relevance for a wide range of hematol. disorders.

=> d l2 bib abs 1-2

L2 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2005:493692 CAPLUS
DN 143:39112
TI Detection of expression level of human dlk gene for diagnosis of liver cancer and the use of anti-Dlk antibody for treatment of cancer
IN Nakamura, Koji; Anzai, Hiroko; Yanai, Hiroyuki; Miyajima, Atsushi
PA Kanagawa Academy of Science and Technology, Japan
SO PCT Int. Appl., 52 pp.
CODEN: PIXXD2
DT Patent
LA Japanese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

PI WO 2005052156 A1 20050609 WO 2004-JP17499 20041125
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
CA 2552553 A1 20050609 CA 2004-2552553 20041125
EP 1702982 A1 20060920 EP 2004-819413 20041125
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK, IS
US 20080112956 A1 20080515 US 2007-580567 20070430
PRAI JP 2003-399331 A 20031128
JP 2003-401585 A 20031201
JP 2003-423237 A 20031219
WO 2004-JP17499 W 20041125
AB This invention provides a method of detecting liver cancer and a novel remedy for cancer having an excellent anticancer effect. The expression of the dlk gene can be assayed by an immunoassay with the use of anti-dlk antibody or an assay of mRNA of the dlk gene.
The remedy for cancer contains, as the active ingredient, an antibody undergoing an antigen-antibody reaction with Dlk expressed on caner cell surface and exhibiting an anticancer effect on the cancer cells.
RE.CNT 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2001:717804 CAPLUS
DN 135:271301
TI Myelodysplastic syndrome diagnosis with Dlk gene expression DNA microarray analysis
IN Aino, Hiroyuki
PA Kirin Brewery Co., Ltd., Japan
SO Jpn. Kokai Tokkyo Koho, 15 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

PI JP 2001269174 A 20011002 JP 2000-85153 20000324
PRAI JP 2000-85153 20000324

AB A method and reagent kits for diagnosis of myelodysplastic syndrome (MDS) using Dlk gene expression as marker, are disclosed. Use of anti -Dlk antibodies for immunodiagnosis and therapy of MDS is claimed. Use of DNA microarrays and 2 dimensional electrophoresis for diagnosis is also claimed. AC133 or CD34 are used as cell surface markers. Myelodysplastic syndrome (MDS) is a slowly progressing hematol. malignancy assocd. with a poor outcome. Despite the relatively high incidence of MDS in the elderly, differentiation of MDS from de novo acute myeloid leukemia (AML) still remains problematic. Identification of genes expressed in an MDS-specific manner would allow the mol. diagnosis of MDS. Toward this goal, AC133 surface marker-pos. hematopoietic stem cell (HSC)-like fractions have been collected from a variety of leukemias in a large-scale and long-term genomics project, referred to as "Blast Bank," and transcriptome of these purified blasts from the patients with MDS were then compared with those from AML through the use of oligonucleotide microarrays. A no. of genes were shown to be expressed in a disease-specific manner either to MDS or AML. Among the former found was the gene encoding the protein Delta-like (Dlk) that is distantly related to the Delta-Notch family of signaling proteins. Because overexpression of Dlk may play a role in the pathogenesis of MDS, the disease specificity of Dlk expression was tested by a quant. "realtime" polymerase chain reaction anal. Examn. of the Blast Bank samples from 22 patients with MDS, 31 with AML, and 8 with chronic myeloid leukemia confirmed the highly selective expression of the Dlk gene in the individuals with MDS. Dlk could be the first candidate mol. to differentiate MDS from AML. The proposal is made that microarray anal. with the Blast Bank samples is an efficient approach to ext. transcriptome data of clin. relevance for a wide range of hematol. disorders.

=> s delta-like
514839 DELTA
474 DELTAS
515077 DELTA
(DELTA OR DELTAS)
903088 LIKE
495 LIKES
903484 LIKE
(LIKE OR LIKES)
L3 935 DELTA-LIKE
(DELTA(W)LIKE)

=> s (delta-like protein)
514839 DELTA

474 DELTAS
515077 DELTA
(DELTA OR DELTAS)
903088 LIKE
495 LIKES
903484 LIKE
(LIKE OR LIKES)
2236592 PROTEIN
1577706 PROTEINS
2612735 PROTEIN
(PROTEIN OR PROTEINS)
L4 34 (DELTA-LIKE PROTEIN)
(DELTA(W)LIKE(W)PROTEIN)

=> s L4 and (liver or hepatocyte or hepatocellular)
603089 LIVER
38646 LIVERS
606355 LIVER
(LIVER OR LIVERS)
54948 HEPATOCYTE
47290 HEPATOCYTES
70587 HEPATOCYTE
(HEPATOCYTE OR HEPATOCYTES)
27774 HEPATOCELLULAR
L5 8 L4 AND (LIVER OR HEPATOCYTE OR HEPATOCELLULAR)

=> d L5 bib abs 1-5

L5 ANSWER 1 OF 8 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2008:534705 CAPLUS
DN 149:511029
TI Drug Insight: antiangiogenic therapies for gastrointestinal cancers-focus on monoclonal antibodies
AU Reinacher-Schick, Anke; Pohl, Michael; Schmiegel, Wolff
CS Department of Medicine, Knappschaftskrankenhaus, Ruhr University Bochum, Bochum, 44892, Germany
SO Nature Clinical Practice Gastroenterology & Hepatology (2008), 5(5), 250-267
CODEN: NCPGAE; ISSN: 1743-4378
PB Nature Publishing Group
DT Journal; General Review
LA English
AB A review. Tumor angiogenesis-the formation of new tumor-assocd. vasculature-has been recognized as an essential event in tumor progression. Vascular endothelial growth factor (VEGF), one of the most important factors involved in tumor angiogenesis, is overexpressed in

several gastrointestinal cancers. In this Review, the authors consider antiangiogenic therapy for the treatment of colorectal, gastric, hepatocellular and pancreatic cancer. Emphasis is placed on the mechanism of action and application of the humanized anti-VEGF monoclonal antibody bevacizumab, but other potential antiangiogenic targets and therapies are also discussed. Tumor angiogenesis is strongly induced by vascular endothelial growth factor (VEGF), which is overexpressed in most human gastrointestinal cancers. VEGF overexpression is known to be assocd. with poor prognosis and survival in patients with various solid tumors. The humanized monoclonal anti-VEGF antibody bevacizumab (Avastin, Genentech Inc., South San Francisco, CA) is a prototypic antiangiogenic compd., and has proven therapeutic benefit combined with conventional chemotherapy-namely, significantly improved progression-free survival in patients with metastatic colorectal cancer. Bevacizumab is the only anti-VEGF antibody that has been approved by the FDA and the European Medicines Agency for the treatment of metastatic colorectal cancer. Several ongoing clin. studies are evaluating the potential of bevacizumab therapy for other gastrointestinal cancers, in combination with chemotherapy, other targeted therapies and/or radiation. Sol. chimeric receptors, tyrosine kinase inhibitors, and monoclonal antibodies against VEGF and mol. targets in the integrin and Delta-like protein 4-Notch pathways are being developed. As tumors acquire resistance to anti-VEGF therapy, further development of antiangiogenic and vascular targets and therapy is warranted.

RE.CNT 144 THERE ARE 144 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 2 OF 8 CAPLUS COPYRIGHT 2008 ACS on STN

AN 2008:344903 CAPLUS

DN 149:398774

TI Delta-like protein (DLK) is a novel immunohistochemical marker for human hepatoblastomas

AU Dezso, Katalin; Halasz, Judit; Bisgaard, Hanne Cathrine; Paku, Sandor; Turanyi, Eszter; Schaff, Zsuzsa; Nagy, Peter

CS First Department of Pathology and Experimental Cancer Research, Semmelweis University, Budapest, 1085, Hung.

SO Virchows Archiv (2008), 452(4), 443-448
CODEN: VARCEM; ISSN: 0945-6317

PB Springer

DT Journal

LA English

AB Delta-like protein (DLK) is a membrane protein with mostly unknown function. It is expressed by several embryonic tissues among others by the hepatoblasts of rodent and human fetal livers. We have investigated in the present study if this

protein is expressed in human hepatoblastomas. The presence of DLK has been studied by std. immunohistochem. in 31 hepatoblastomas and in several differential diagnostically related tumors: hepatocellular carcinomas and in undifferentiated childhood neoplasms. All the hepatoblastomas were pos. for DLK; the surrounding liver tissue remained neg. The reaction was present in the epithelial component of the tumors. The staining pattern was mostly membranous, occasionally cytoplasmic. The other studied tumors were neg. for DLK, except one hepatocellular carcinoma and the differentiating cells of two ganglioneuroblastomas. Therefore, DLK seems to be a highly sensitive and specific marker for hepatoblastomas.

RE.CNT 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2008 ACS on STN

AN 2007:792684 CAPLUS

DN 148:140366

TI Remarkable heterogeneity displayed by oval cells in rat and mouse models of stem cell-mediated liver regeneration

AU Jelnes, Peter; Satoni-Rugiu, Eric; Rasmussen, Morten; Friis, Susanne Lunoee; Nielsen, Jens Hoeriis; Tygstrup, Niels; Bisgaard, Hanne Cathrine

CS Danish Stem Cell Research Centre, Department of Cellular and Molecular Medicine, The Panum Institute, University of Copenhagen, Copenhagen, Den.

SO Hepatology (Hoboken, NJ, United States) (2007), 45(6), 1462-1470 CODEN: HPTLD9; ISSN: 0270-9139

PB John Wiley & Sons, Inc.

DT Journal

LA English

AB The exptl. protocols used in the investigation of stem cell-mediated liver regeneration in rodents are characterized by activation of the hepatic stem cell compartment in the canals of Hering followed by transit amplification of oval cells and their subsequent differentiation along hepatic lineages. Although the protocols are numerous and often used interchangeably across species, a thorough comparative phenotypic anal. of oval cells in rats and mice using well-established and generally acknowledged mol. markers has not been provided. In the present study, we evaluated and compared the mol. phenotypes of oval cells in several of the most commonly used protocols of stem cell-mediated liver regeneration-namely, treatment with 2-acetylaminofluorene and partial (70%) hepatectomy (AAF/PHx); a choline-deficient, ethionine-supplemented (CDE) diet; a 3,5-diethoxycarbonyl-1,4-dihydro-collidin (DDC) diet; and N-acetyl-paraaminophen (APAP). Reproducibly, oval cells showing reactivity for cytokeratins (CKs), muscle pyruvate kinase (MPK), the ATP-binding cassette transporter ABCG2/BCRP1 (ABCG2), alpha-fetoprotein (AFP), and delta-like protein 1/preadipocyte

factor 1 (Dlk/Pref-1) were induced in rat liver treated according to the AAF/PHx and CDE but not the DDC protocol. In mouse liver, the CDE, DDC, and APAP protocols all induced CKs and ABCG2-pos. oval cells. However, AFP and Dlk/Pref-1 expression was rarely detected in oval cells. Conclusion: Our results delineate remarkable phenotypic discrepancies exhibited by oval cells in stem cell-mediated liver regeneration between rats and mice and underline the importance of careful extrapolation between individual species.

RE.CNT 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 4 OF 8 CAPLUS COPYRIGHT 2008 ACS on STN

AN 2004:353594 CAPLUS

DN 141:138260

TI Transit-amplifying ductular (oval) cells and their hepatocytic progeny are characterized by a novel and distinctive expression of delta-like protein/preadipocyte factor 1/fetal antigen 1

AU Jensen, Charlotte Harken; Jauho, Eva Irene; Santoni-Rugiu, Eric; Holmskov, Uffe; Teisner, Borge; Tygstrup, Niels; Bisgaard, Hanne Cathrine

CS Department of Immunology and Microbiology, Danish Stem Cell Research Center, University of Southern Denmark, Odense, Den.

SO American Journal of Pathology (2004), 164(4), 1347-1359
CODEN: AJPAA4; ISSN: 0002-9440

PB American Society for Investigative Pathology

DT Journal

LA English

AB Hepatic regeneration from toxic or surgical injury to the adult mammalian liver, endorses different cellular responses within the hepatic lineage. The mol. mechanisms detg. commitment of a cell population at a specific lineage level to participate in liver repair as well as the fate of its progeny in the hostile environment created by the injury are not well defined. Based on the role of the Notch/Delta/Jagged system in cell fate specification and recent reports linking Notch signaling with normal bile duct formation in mouse and human liver, we examd. the expression of Notch1, Notch2, Notch3, Delta1, Delta3, Jagged1, and Jagged2, and delta-like protein/preadipocyte factor 1/fetal antigen 1 (dlk) in four well-defined exptl. rat models of liver injury and regeneration. Although Delta3 and Jagged2 were undetectable by reverse transcriptase-polymerase chain reaction and Northern blot, we obsd. the most significant up-regulation of all other transcripts in the 2-acetylaminofluorene-70% hepatectomy (AAF/PHx) model, in which liver mass is restored by proliferation and differentiation of transit-amplifying ductular (oval) cells. The most profound change was obsd. for dlk. Accordingly, immunohistochem. analyses in the AAF/PHx model showed a specific expression of dlk in atypical

ductular structures composed of oval cells. Delta-like protein was not obsd. in proliferating hepatocytes or bile duct cells after partial hepatectomy or ligation of the common bile duct whereas clusters of dlk immunoreactive oval cells were found in both the retrorsine and the AAF/PHx models. Finally, we used dlk to isolate .alpha.-fetoprotein-pos. cells from fetal and adult regenerating rat liver by a novel antibody panning technique.

RE.CNT 63 THERE ARE 63 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 5 OF 8 CAPLUS COPYRIGHT 2008 ACS on STN

AN 2003:688933 CAPLUS

DN 139:210402

TI Use of delta-like protein to inhibit the differentiation of stem cells

IN Witte, Larry; Pytowski, Bronislaw; Moore, Kateri A.; Lemischka, Ihor R.

PA ImClone Systems Incorporated, USA; Trustees of Princeton University

SO U.S., 13 pp., Cont.-in-part of U.S. Ser. No. 612,719, abandoned.
CODEN: USXXAM

DT Patent

LA English

FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 6613565	B1	20030902	US 1998-142027	19981208
WO 9731647	A1	19970904	WO 1997-US3520	19970303

W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN

RW: GH, KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

PRAI US 1996-609533 B2 19960301
US 1996-612719 B2 19960308
WO 1997-US3520 W 19970303

AB Primitive hematopoietic stem cells are closely assocd. with discrete in vivo microenvironments. These "niches" are thought to provide the mol. signals that mediate stem cell differentiation and self renewal. We have dissected the fetal liver microenvironment into distinct cellular components by establishing an extensive panel of stromal cell lines. One particular cell line maintains repopulating stem cells for prolonged in vitro culture periods. A subtraction cloning strategy has yielded a cDNA which encodes a cell surface glycoprotein with a restricted pattern of expression among stromal cell lines. This mol., previously

identified as dlk/Pref-1, contains EGF-like repeats which are related to those in the Notch/Delta/Serrate family of proteins. We have investigated the potential role of this mol. in hematopoietic stem/progenitor cell regulation. We show that the dlk protein displays activity on purified stem cells by promoting the formation of "cobblestone areas" of proliferation. These cobblestone areas contain both primitive high-proliferative potential progenitors as well as in vivo repopulating stem cells.

RE.CNT 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s (dlk or Pref-1 or (preadipocyte factor 1) or (fetal antigen 1))

198 DLK
1 DLKS
199 DLK
(DLK OR DLKS)
606 PREF
40 PREFS
641 PREF
(PREF OR PREFS)
9907870 1
122 PREF-1
(PREF(W)1)
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2174 PREADIPOCYTES
3198 PREADIPOCYTE
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1064477 FACTORS
1842708 FACTOR
(FACTOR OR FACTORS)
9907870 1
70 PREADIPOCYTE FACTOR 1
(PREADIPOCYTE(W)FACTOR(W)1)
97065 FETAL
9 FETALS
97072 FETAL
(FETAL OR FETALS)
349485 ANTIGEN
274476 ANTIGENS
441215 ANTIGEN
(ANTIGEN OR ANTIGENS)
9907870 1
31 FETAL ANTIGEN 1

(FETAL(W)ANTIGEN(W)1)
L6 332 (DLK OR PREF-1 OR (PREADIPOCYTE FACTOR 1) OR (FETAL ANTIGEN 1))

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38646 LIVERS
606355 LIVER
(LIVER OR LIVERS)
40950 HEPATOMA
2990 HEPATOMAS
41751 HEPATOMA
(HEPATOMA OR HEPATOMAS)
54948 HEPATOCYTE
47290 HEPATOCYTES
70587 HEPATOCYTE
(HEPATOCYTE OR HEPATOCYTES)
27774 HEPATOCELLULAR
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PROCESSING COMPLETED FOR L7
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=> s L8 and (cancer or carcinoma or tumor or tumour or malignancy or maligant or neoplasia)
L9 40 S L8
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56511 CANCERS
398052 CANCER
(CANCER OR CANCERS)
190620 CARCINOMA
35947 CARCINOMAS
173 CARCINOMATA
199212 CARCINOMA
(CARCINOMA OR CARCINOMAS OR CARCINOMATA)
475868 TUMOR
175988 TUMORS
529851 TUMOR
(TUMOR OR TUMORS)
3993 TUMOUR
1500 TUMOURS
5397 TUMOUR
(TUMOUR OR TUMOURS)
19134 MALIGNANCY

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      35845 MALIGNANCY
            (MALIGNANCY OR MALIGNANCIES)
         10 MALIGANT
      16221 NEOPLASIA
       1616 NEOPLASIAS
      17436 NEOPLASIA
            (NEOPLASIA OR NEOPLASIAS)
L10       13 L9 AND (CANCER OR CARCINOMA OR TUMOR OR TUMOUR OR
MALIGNANCY OR
          MALIGANT OR NEOPLASIA)

=> s L10 and antibody
      339332 ANTIBODY
      408617 ANTIBODIES
      540130 ANTIBODY
            (ANTIBODY OR ANTIBODIES)
L11        4 L10 AND ANTIBODY

=> d L11 bib abs 1-4

L11  ANSWER 1 OF 4  CAPLUS  COPYRIGHT 2008 ACS on STN
AN   2008:585336  CAPLUS
DN   148:536036
TI   Humainzed anti-human Dlk-1 antibodies and conjugates
     for cancer diagnosis and therapy
IN   Nakamura, Koji; Tajima, Rie
PA   Livtech Inc., Japan
SO   PCT Int. Appl., 112pp.
     CODEN: PIXXD2
DT   Patent
LA   Japanese
FAN.CNT 1
     PATENT NO.         KIND   DATE       APPLICATION NO.      DATE
     ---------------    ----   --------   -------------------  --------
PI   WO 2008056833      A1     20080515   WO 2007-JP72335      20071112
        W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA,
          CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI,
          GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG,
          KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME,
          MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL,
          PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN,
          TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
        RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,
          IS, IT, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, BF,
          BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW,
```

GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRAI JP 2006-305355 A 20061110

AB Disclosed are: an antibody capable of reacting specifically with hDlk-1 and shows an anti-tumor activity in vivo (an anti-hDlk-1 antibody); a fragment of the antibody; a hybridoma capable of producing the antibody; a complex of the antibody or a fragment thereof and a physiol. active substance; a pharmaceutical compn., a therapeutic agent for a tumor, a tumor angiogenesis inhibitor or a diagnostic agent for a tumor, which comprises the antibody or the like; a method for the detection of a tumor; a kit for the detection and/or diagnosis of a tumor; and others.

RE.CNT 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2008 ACS on STN

AN 2005:1240740 CAPLUS

DN 144:4118

TI Genes showing changes in expression in developing and aging in mouse muscle for use in diagnosis and treatment of disease

IN Kopchick, John J.; Coschigano, Karen T.; Boyce, Keith S.; Kriete, Andres

PA Ohio University, USA; Icoria, Inc.

SO PCT Int. Appl., 440 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2005110460	A2	20051124	WO 2005-US14441	20050428
WO 2005110460	A3	20060413		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI US 2004-566068P P 20040429

US 2004-577930P P 20040609

AB Mouse genes that show changes in levels of expression in muscle are identified. These genes, and their human equiv., may be useful as targets in the control of aging and in the treatment of diseases assocd. with accelerated aging (no data.). The human mols. may also be used as markers of biol. aging.

L11 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2005:493692 CAPLUS
DN 143:39112
TI Detection of expression level of human dlk gene for diagnosis of liver cancer and the use of anti-Dlk antibody for treatment of cancer
IN Nakamura, Koji; Anzai, Hiroko; Yanai, Hiroyuki; Miyajima, Atsushi
PA Kanagawa Academy of Science and Technology, Japan
SO PCT Int. Appl., 52 pp.
CODEN: PIXXD2
DT Patent
LA Japanese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2005052156	A1	20050609	WO 2004-JP17499	20041125
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2552553	A1	20050609	CA 2004-2552553	20041125
EP 1702982	A1	20060920	EP 2004-819413	20041125
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK, IS				
US 20080112956	A1	20080515	US 2007-580567	20070430
PRAI JP 2003-399331	A	20031128		
JP 2003-401585	A	20031201		
JP 2003-423237	A	20031219		
WO 2004-JP17499	W	20041125		

AB This invention provides a method of detecting liver cancer and a novel remedy for cancer having an excellent anticancer effect. The expression of the dlk gene can be

assayed by an immunoassay with the use of anti-dlk antibody or an assay of mRNA of the dlk gene. The remedy for cancer contains, as the active ingredient, an antibody undergoing an antigen-antibody reaction with Dlk expressed on caner cell surface and exhibiting an anticancer effect on the cancer cells.

RE.CNT 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2002:429201 CAPLUS
DN 137:4997
TI Method for diagnosing allergic diseases using DNA and protein microarray technology
IN Schmidt-Weber, Carsten; Blaser, Kurt; Wohlfahrt, Jan
PA Genescan Europe Ag, Germany
SO PCT Int. Appl., 61 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002044732	A2	20020606	WO 2001-EP13937	20011129
WO 2002044732	A3	20030327		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

EP 1221618	A1	20020710	EP 2000-126117	20001129

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR

AU 2002021906	A	20020611	AU 2002-21906	20011129
PRAI EP 2000-126117	A	20001129		
WO 2001-EP13937	W	20011129		

AB MRNA of activated lymphocytes such as CD4+ T cells allows differential diagnosis of allergic diseases. The CD4+ T cells are isolated and stimulated under defined conditions in vitro. Subsequently, mRNA is subjected to multigene anal. such as DNA arrays. Expression profiling

images, such as gene expression profiles, can be created, which allow on the basis of the activated T cell mRNA the prediction of certain phenotypes such as asthma or atopic dermatitis.

=> d L10 bib abs 1-13

L10 ANSWER 1 OF 13 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2008:585336 CAPLUS
DN 148:536036
TI Humainzed anti-human Dlk-1 antibodies and conjugates for cancer diagnosis and therapy
IN Nakamura, Koji; Tajima, Rie
PA Livtech Inc., Japan
SO PCT Int. Appl., 112pp.
CODEN: PIXXD2
DT Patent
LA Japanese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2008056833	A1	20080515	WO 2007-JP72335	20071112

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRAI JP 2006-305355 A 20061110
AB Disclosed are: an antibody capable of reacting specifically with hDlk-1 and shows an anti-tumor activity in vivo (an anti-hDlk-1 antibody); a fragment of the antibody; a hybridoma capable of producing the antibody; a complex of the antibody or a fragment thereof and a physiol. active substance; a pharmaceutical compn., a therapeutic agent for a tumor, a tumor angiogenesis inhibitor or a diagnostic agent for a tumor, which comprises the antibody or the like; a method for the detection of a tumor; a kit for the detection and/or diagnosis of a tumor; and others.
RE.CNT 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 2 OF 13 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2008:535338 CAPLUS
DN 149:6982
TI Adipogenic capacity and the susceptibility to type 2 diabetes and metabolic syndrome
AU Wang, May-Yun; Grayburn, Paul; Chen, Shuyuan; Ravazzola, Mariella; Orci, Lelio; Unger, Roger H.
CS Touchstone Center for Diabetes Research, University of Texas Southwestern Medical Center, Dallas, TX, 75390-8854, USA
SO Proceedings of the National Academy of Sciences of the United States of America (2008), 105(16), 6139-6144
CODEN: PNASA6; ISSN: 0027-8424
PB National Academy of Sciences
DT Journal
LA English
AB To det. whether adipocyte storage capacity influences the onset and severity of type 2 diabetes and other components of the metabolic syndrome, we made normal and db/db mice resistant to obesity by overexpressing leptin receptor-b on the aP2-Lepr-b promoter. On a 4% diet, these mice have no phenotype, but on a 60% fat diet, they resist diet-induced obesity because constitutive adipocyte-specific overexpression of Lepr-b prevents obesity via the antilipogenic autocrine/paracrine action of leptin on adipocytes. After 8 mo on the same 60% fat diet, body fat of transgenic mice was 70% below WT controls. Cardiac and liver fat was elevated in the transgenics, and their hyperinsulinemia was more marked, suggesting greater insulin resistance. The aP2-Lepr-b transgene also prevented obesity in db/db mice; at 10 wk of age their body fat was half that of the db/db mice. This lack of obesity was attributable to reduced expression of sterol regulatory element binding protein-1c and its target lipogenic enzymes in adipose tissue and a 6-fold increase in Pref-1 mRNA. Severe diabetes was present in transgenics at 4 wk of age, 10 wk before db/db controls. Echocardiog. evidence of cardiomyopathy appeared at 10 wk, weeks before the db/db mice. Histol., loss of .beta. cells and myocardial fibrosis was present in the transgenic group at least 6 wk before the db/db mice. These results suggest that the expression level of genes that regulate the adipogenic response to overnutrition profoundly influences the age of onset and severity of diet-induced type 2 diabetes and co-morbidities.
RE.CNT 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 3 OF 13 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2008:344903 CAPLUS

DN 149:398774
TI Delta-like protein (DLK) is a novel immunohistochemical marker for human hepatoblastomas
AU Dezso, Katalin; Halasz, Judit; Bisgaard, Hanne Cathrine; Paku, Sandor; Turanyi, Eszter; Schaff, Zsuzsa; Nagy, Peter
CS First Department of Pathology and Experimental Cancer Research, Semmelweis University, Budapest, 1085, Hung.
SO Virchows Archiv (2008), 452(4), 443-448
CODEN: VARCEM; ISSN: 0945-6317
PB Springer
DT Journal
LA English
AB Delta-like protein (DLK) is a membrane protein with mostly unknown function. It is expressed by several embryonic tissues among others by the hepatoblasts of rodent and human fetal livers. We have investigated in the present study if this protein is expressed in human hepatoblastomas. The presence of DLK has been studied by std. immunohistochem. in 31 hepatoblastomas and in several differential diagnostically related tumors: hepatocellular carcinomas and in undifferentiated childhood neoplasms. All the hepatoblastomas were pos. for DLK; the surrounding liver tissue remained neg. The reaction was present in the epithelial component of the tumors. The staining pattern was mostly membranous, occasionally cytoplasmic. The other studied tumors were neg. for DLK, except one hepatocellular carcinoma and the differentiating cells of two ganglioneuroblastomas. Therefore, DLK seems to be a highly sensitive and specific marker for hepatoblastomas.
RE.CNT 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 4 OF 13 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2007:1183527 CAPLUS
DN 148:7756
TI Mice heterozygous for tumor necrosis factor-.alpha. converting enzyme are protected from obesity-induced insulin resistance and diabetes
AU Serino, Matteo; Menghini, Rossella; Fiorentino, Loredana; Amoruso, Roberta; Mauriello, Alessandro; Lauro, Davide; Sbraccia, Paolo; Hribal, Marta L.; Lauro, Renato; Federici, Massimo
CS Laboratory of Molecular Medicine, Department of Internal Medicine, University of Rome "Tor Vergata", Rome, Italy
SO Diabetes (2007), 56(10), 2541-2546
CODEN: DIAEAZ; ISSN: 0012-1797
PB American Diabetes Association, Inc.
DT Journal

LA English
AB Tumor necrosis factor (TNF)-.alpha. is known to affect insulin sensitivity, glucose, and lipid metab. through alternative and redundant mechanisms at both translational and post-translational levels. TNF-.alpha. exerts its paracrine effects once the membrane-anchored form is shed and released from the cell membrane. TNF-.alpha. cleavage is regulated by TNF-.alpha. converting enzyme (TACE), which regulates the function of several transmembrane proteins, such as interleukin-6 receptor and epidermal growth factor receptor ligands. The role of TACE in high-fat diet (HFD)-induced obesity and its metabolic complications is unknown. To gain insights into the role of TACE in metabolic disorders, we used Tace+/- mice fed a std. or high-fat diet for 16 wk. We obsd. that Tace+/- mice are relatively protected from obesity and insulin resistance compared with wild-type littermates. When fed an HFD, wild-type mice exhibited visceral obesity, increased free fatty acid and monocyte chemo-attractant protein (MCP)1 levels, hypoadiponectinemia, glucose intolerance, and insulin resistance compared with Tace+/- mice. Interestingly, Tace+/- mice exhibited increased uncoupling protein-1 and GLUT4 expression in white adipose tissue. The results suggest that modulation of TACE activity is a new pathway to be investigated for development of agents acting against obesity and its metabolic complications.
RE.CNT 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 5 OF 13 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2006:498140 CAPLUS
DN 145:77714
TI Protein and cDNA sequences of human liver cancer -related gene DLK1 (delta like 1 homolog) and their uses in diagnosis and treatment of liver cancer
IN Huang, Jian; Zhang, Xin; Han, Zeguang
PA Shanghai Human Genome Research Center, Peop. Rep. China
SO Faming Zhuanli Shenqing Gongkai Shuomingshu, 20 pp.
CODEN: CNXXEV
DT Patent
LA Chinese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI CN 1714862	A	20060104	CN 2004-10025561	20040629
CN 1304046	C	20070314		
PRAI CN 2004-10025561		20040629		

AB Described are the protein and cDNA sequences of human liver cancer-related gene DLK1 (delta like 1 homolog). Gene DLK1 and

its coded protein are useful in diagnosis and treatment of primary liver cancer. Gene DLK1 locates at chromosome 14q32, and its expression in liver cancer tissue is significantly higher than that in adjacent liver tissue. DLK1 was highly expressed in placenta and also expressed in heart and skeletal muscle. DLK1 was expressed in liver tumor cell line Hep3B, HepG2, and Huh-7. The invention also provides a test kit and a biochip for diagnosis of liver cancer.

L10 ANSWER 6 OF 13 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2005:1240740 CAPLUS
DN 144:4118
TI Genes showing changes in expression in developing and aging in mouse muscle for use in diagnosis and treatment of disease
IN Kopchick, John J.; Coschigano, Karen T.; Boyce, Keith S.; Kriete, Andres
PA Ohio University, USA; Icoria, Inc.
SO PCT Int. Appl., 440 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2005110460	A2	20051124	WO 2005-US14441	20050428
WO 2005110460	A3	20060413		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI US 2004-566068P P 20040429
US 2004-577930P P 20040609

AB Mouse genes that show changes in levels of expression in muscle are identified. These genes, and their human equiv., may be useful as targets in the control of aging and in the treatment of diseases assocd. with accelerated aging (no data.). The human mols. may also be used as markers of biol. aging.

L10 ANSWER 7 OF 13 CAPLUS COPYRIGHT 2008 ACS on STN

AN 2005:493692 CAPLUS
DN 143:39112
TI Detection of expression level of human dlk gene for diagnosis of liver cancer and the use of anti-Dlk antibody for treatment of cancer
IN Nakamura, Koji; Anzai, Hiroko; Yanai, Hiroyuki; Miyajima, Atsushi
PA Kanagawa Academy of Science and Technology, Japan
SO PCT Int. Appl., 52 pp.
CODEN: PIXXD2
DT Patent
LA Japanese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2005052156	A1	20050609	WO 2004-JP17499	20041125
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2552553	A1	20050609	CA 2004-2552553	20041125
EP 1702982	A1	20060920	EP 2004-819413	20041125
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK, IS				
US 20080112956	A1	20080515	US 2007-580567	20070430
PRAI JP 2003-399331	A	20031128		
JP 2003-401585	A	20031201		
JP 2003-423237	A	20031219		
WO 2004-JP17499	W	20041125		

AB This invention provides a method of detecting liver cancer and a novel remedy for cancer having an excellent anticancer effect. The expression of the dlk gene can be assayed by an immunoassay with the use of anti-dlk antibody or an assay of mRNA of the dlk gene. The remedy for cancer contains, as the active ingredient, an antibody undergoing an antigen-antibody reaction with Dlk expressed on caner cell surface and exhibiting an anticancer effect on the cancer cells.
RE.CNT 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 8 OF 13 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2004:544587 CAPLUS
DN 141:185521
TI Mixed lineage kinase 3 (MLK3)-activated p38 MAP kinase mediates transforming growth factor-.beta.-induced apoptosis in hepatoma cells
AU Kim, Ki-Yong; Kim, Byung-Chul; Xu, Zhiheng; Kim, Seong-Jin
CS Laboratory of Cell Regulation and Carcinogenesis, NCI, National Institutes of Health, Bethesda, MD, 20892, USA
SO Journal of Biological Chemistry (2004), 279(28), 29478-29484
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
AB Although transforming growth factor .beta.1 (TGF-.beta.1) acts via the Smad signaling pathway to initiate de novo gene transcription, the TGF-.beta.1-induced MAPK kinase activation that is involved in the regulation of apoptosis is less well understood. Even though the p38 MAP kinase and c-Jun N-terminal kinases (JNKs) are involved in TGF-.beta.1-induced cell death in hepatoma cells, the upstream mediators of these kinases remain to be defined. The authors show here that the members of the mixed lineage kinase (MLK) family (including MLK1, MLK2, MLK3, and dual leucine zipper-bearing kinase (DLK)) are expressed in FaO rat hepatoma cells and are likely to act between p38 and TGF-.beta. receptor kinase in death signaling. TGF-.beta.1 treatment leads to an increase in MLK3 activity. Overexpression of MLK3 enhances TGF-.beta.1-induced apoptotic death in FaO cells and Hep3B human hepatoma cells, whereas expression of the dominant-neg. forms of MLK3 suppresses cell death induced by TGF-.beta.1. The dominant-neg. forms of MLK1 and -2 also suppress TGF-.beta.1-induced cell death. In MLK3-overexpressing cells, ERK, JNKs, and p38 MAP kinases were further activated in response to TGF-.beta.1 compared with the control cells. In contrast, overexpression of the dominant-neg. MLK3 resulted in suppression of TGF-.beta.1-induced MAP kinase activation and TGF-.beta.1-induced caspase-3 activation. The authors also show that only the inhibition of the p38 pathway suppressed TGF-.beta.1-induced apoptosis. These observations support a role for MLKs in the TGF-.beta.1-induced cell death mechanism.
RE.CNT 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 9 OF 13 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2004:449883 CAPLUS
DN 140:402911

TI Binary prediction tree modeling with many predictors and its uses in clinical and genomic applications

IN Nevins, Joseph R.; West, Mike; Huang, Andrew T.

PA Duke University, USA

SO PCT Int. Appl., 886 pp.
CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 5

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2004038376	A2	20040506	WO 2003-XA33946	20031024
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
WO 2004038376	A2	20040506	WO 2003-US33946	20031024
WO 2004038376	A3	20040826		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
PRAI US 2002-420729P	P	20021024		
US 2002-421062P	P	20021025		
US 2002-421102P	P	20021025		
US 2002-424701P	P	20021108		
US 2002-424715P	P	20021108		
US 2002-424718P	P	20021108		
US 2002-425256P	P	20021112		
US 2003-448461P	P	20030221		
US 2003-448462P	P	20030221		
US 2003-457877P	P	20030327		
US 2003-458373P	P	20030331		
WO 2003-US33946	A	20031024		

AB The statistical anal. described and claimed is a predictive statistical tree model that overcomes several problems obsd. in prior statistical models and regression analyses, while ensuring greater accuracy and predictive capabilities. Although the claimed use of the predictive statistical tree model described herein is directed to the prediction of a disease in individuals, the claimed model can be used for a variety of applications including the prediction of disease states, susceptibility of disease states or any other biol. state of interest, as well as other applicable non-biol. states of interest. This model first screens genes to reduce noise, applies kmeans correlation-based clustering targeting a large no. of clusters, and then uses singular value decompns. (SVD) to ext. the single dominant factor (principal component) from each cluster. This generates a statistically significant no. of cluster-derived singular factors, that are referred to as metagenes, that characterize multiple patterns of expression of the genes across samples. The strategy aims to ext. multiple such patterns while reducing dimension and smoothing out gene-specific noise through the aggregation within clusters. Formal predictive anal. then uses these metagenes in a Bayesian classification tree anal. This generates multiple recursive partitions of the sample into subgroups (the 'leaves' of the classification tree), and assocs. Bayesian predictive probabilities of outcomes with each subgroup. Overall predictions for an individual sample are then generated by averaging predictions, with appropriate wts., across many such tree models. The model includes the use of iterative out-of-sample, cross-validation predictions leaving each sample out of the data set one at a time, refitting the model from the remaining samples and using it to predict the hold-out case. This rigorously tests the predictive value of a model and mirrors the real-world prognostic context where prediction of new cases as they arise is the major goal.

L10 ANSWER 10 OF 13 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2002:429201 CAPLUS
DN 137:4997
TI Method for diagnosing allergic diseases using DNA and protein microarray technology
IN Schmidt-Weber, Carsten; Blaser, Kurt; Wohlfahrt, Jan
PA Genescan Europe Ag, Germany
SO PCT Int. Appl., 61 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002044732	A2	20020606	WO 2001-EP13937	20011129
WO 2002044732	A3	20030327		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

EP 1221618 A1 20020710 EP 2000-126117 20001129

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR

AU 2002021906 A 20020611 AU 2002-21906 20011129

PRAI EP 2000-126117 A 20001129

WO 2001-EP13937 W 20011129

AB MRNA of activated lymphocytes such as CD4+ T cells allows differential diagnosis of allergic diseases. The CD4+ T cells are isolated and stimulated under defined conditions in vitro. Subsequently, mRNA is subjected to multigene anal. such as DNA arrays. Expression profiling images, such as gene expression profiles, can be created, which allow on the basis of the activated T cell mRNA the prediction of certain phenotypes such as asthma or atopic dermatitis.

L10 ANSWER 11 OF 13 CAPLUS COPYRIGHT 2008 ACS on STN

AN 2002:391912 CAPLUS

DN 137:1836

TI Measurement of DNA methylation for analysis of the toxicology of substances

IN Olek, Alexander; Piepenbrock, Christian; Berlin, Kurt

PA Epigenomics Ag, Germany

SO PCT Int. Appl., 113 pp.

CODEN: PIXXD2

DT Patent

LA German

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002040710	A2	20020523	WO 2001-EP12951	20011108
WO 2002040710	A3	20030530		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

DE 10056802 A1 20020529 DE 2000-10056802 20001114

DE 10056802 B4 20050616

AU 2002023672 A 20020527 AU 2002-23672 20011108

EP 1337668 A2 20030827 EP 2001-996625 20011108

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR

JP 2004513650 T 20040513 JP 2002-543021 20011108

US 20040048279 A1 20040311 US 2003-416905 20030514

PRAI DE 2000-10056802 A 20001114

WO 2001-EP12951 W 20011108

AB The invention relates to a method for anal. of the toxicol. of a substance by measuring its effects using changes in DNA methylation as an indicator of toxicol. According to the invention, a DNA sample is taken from an organism or a cell culture which has been exposed to a specific substance which is to be examd. on account of its toxicol. effect. The DNA contained in said sample is chem. pre-treated and the base sequence of a section of the modified DNA is detd. The preferred method is to convert cytosine in CpG dinucleotides to uracil using bisulfite. Probes specific for cytosine- or uracil-contg. DNA can be used to detect changes in methylation. From there, a characteristic methylation state or a characteristic methylation model is detd. for the sample. By comparison with data from methylation states of other samples, the effect of a substance on the organism or the cell culture is detd. and/or compared to other substances in toxicol. terms. A panel of sequences that can be used to analyze the effects of poisons is described.

L10 ANSWER 12 OF 13 CAPLUS COPYRIGHT 2008 ACS on STN

AN 2000:314864 CAPLUS

DN 132:344076

TI Method for detecting endocrine disruptor-responsive genes and for screening endocrine disruptors

IN Kondo, Akihiro; Sagawa, Hiroaki; Mineno, Junichi; Kimizuka, Fusao; Kato, Ikunoshin

PA Takara Shuzo Co., Ltd., Japan

SO PCT Int. Appl., 81 pp.

CODEN: PIXXD2

DT Patent

LA Japanese

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000026404	A1	20000511	WO 1999-JP5964	19991028

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

AU 9964878 A 20000522 AU 1999-64878 19991028
EP 1126035 A1 20010822 EP 1999-952794 19991028
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO
PRAI JP 1998-310285 A 19981030
WO 1999-JP5964 W 19991028

AB A method and compns. for detecting genes affected by endocrine-disrupting chems. and for identifying endocrine-disrupting chems. are claimed. The method comprises prepg. a nucleic acid sample contg. mRNAs or cDNAs originating in cells, tissues, or organisms which have been brought into contact with a sample contg. the endocrine disruptor. The nucleic acid sample is hybridized with DNA arrays wherein genes which might be affected by the endocrine disruptor or DNA fragments originating in these genes have been fixed. The results obtained are then compared with the results obtained with the control sample to select the gene affected by the endocrine disruptor. Endocrine disruptors are selected from dioxins, org. chloro compds., phenols, futalic acid esters, arom. hydrocarbons, agrochems., org. tin compds., and estrogens, among others. The effect of 3 chems., 17-.beta. estradiol (E2), diethylstilbestrol (DES), and bisphenol A (BisA) on 33 candidate genes belonging to the categories of nuclear receptor/nuclear receptor transcriptional coupling, kinase-type signal transducer, gonad differentiation factor, oncogene, and receptor-type kinase, were examd. by the method of this invention. Expression of most of the genes was either increased or decreased by exposure to these chems.

RE.CNT 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 13 OF 13 CAPLUS COPYRIGHT 2008 ACS on STN
AN 1998:740427 CAPLUS
DN 130:93929
TI Insulin resistance and diabetes mellitus in transgenic mice expressing nuclear SREBP-1c in adipose tissue: model for congenital generalized lipodystrophy
AU Shimomura, Iichiro; Hammer, Robert E.; Richardson, James A.; Ikemoto, Shinji; Bashmakov, Yuriy; Goldstein, Joseph L.; Brown, Michael S.
CS Department of Molecular Genetics, The University of Texas Southwestern

Medical Center at Dallas, Dallas, TX, 75235, USA
SO Genes & Development (1998), 12(20), 3182-3194
CODEN: GEDEEP; ISSN: 0890-9369
PB Cold Spring Harbor Laboratory Press
DT Journal
LA English
AB Overexpression of the nuclear form of sterol regulatory element-binding protein-1c (nSREBP-1c/ADD1) in cultured 3T3-L1 preadipocytes was shown previously to promote adipocyte differentiation. Here, we produced transgenic mice that overexpress nSREBP-1c in adipose tissue under the control of the adipocyte-specific aP2 enhancer/promoter. A syndrome with the following features was obsd.: (1) Disordered differentiation of adipose tissue. White fat failed to differentiate fully, and the size of white fat depots was markedly decreased. Brown fat was hypertrophic and contained fat-laden cells resembling immature white fat. Levels of mRNA encoding adipocyte differentiation markers (C/EBP.alpha., PPAR.gamma., adipsin, leptin, UCP1) were reduced, but levels of Pref-1 and TNF.alpha. were increased. (2) Marked insulin resistance with 60-fold elevation in plasma insulin. (3) Diabetes mellitus with elevated blood glucose (>300 mg/dL) that failed to decline when insulin was injected. (4) Fatty liver from birth and elevated plasma triglyceride levels later in life. These mice exhibit many of the features of congenital generalized lipodystrophy (CGL), an autosomal recessive disorder in humans.
RE.CNT 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT